

Abnormal visual connectivity in Eyelid Myoclonia with Absences: evidences from Electrocortical Connectivity and Non-linear Quantitative Analysis of EEG Signal

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Objectives

Eyelid Myoclonia with Absences (EMA) is an epileptic syndrome characterized by eyelid myoclonia with or without absences, eye closure-induced EEG paroxysms and photosensitivity. Pathophysiological mechanisms of visual sensitivity in EMA are not-fully understood. The main objective of our study was to analyze, by means of low resolution electromagnetic tomography (LORETA) and EEG quantitative analysis with a nonlinear methodology, the electrocortical networks possibly implicated in the visual sensitivity in a group of patients with EMA.

Methods

We analyzed data of 10 subjects with EMA and of 10 control subjects. Four seconds artefacts-free EEG signal epochs recorded from 19 electrodes placed according to the 10-20 system were analyzed, during resting state, eyes opened and eyes-closed tasks.

EEG networks have been computed using independent components analysis (ICA) LORETA.

The power law exponent β was obtained for each coordinate as minus the slope of the power spectrum versus frequency in a Log-Log scale, to estimate self-similarity of the electrocortical signal. β values ~ 1 imply self-similarity, typical property of fractal phenomena.

Results

Reduction of alpha activity over the lingual gyrus in patients compared to controls during the resting state.



alpha

Reduction of beta activity over the middle frontal gyrus in patients compared to controls during the resting state.

Increase of alpha activity over the precuneus in patients compared to controls during the resting state.



alpha

Increase of beta activity over the inferior frontal gyrus in patients compared to controls during the eyes-closed task.



Differences in the values of β index between patients and controls during the resting state.

Differences in the values of β index in the group of patients between the resting state and the eyes-closed task.



Conclusions

The findings of our study seem to confirm the role of an intrinsic abnormality of the occipital cortex with an altered occipital-frontal network in determining the visual sensitivity in EMA.



